CHAPTER 11

LETHAL GIFT OF LIVESTOCK

WE HAVE NOW TRACED HOW FOOD PRODUCTION AROSE in a few centers, and how it spread at unequal rates from there to other areas. Those geographic differences constitute important ultimate answers to Yali's question about why different peoples ended up with disparate degrees of power and affluence. However, food production itself is not a proximate cause. In a one-on-one fight, a naked farmer would have no advantage over a naked hunter-gatherer.

Instead, one part of the explanation for farmer power lies in the much denser populations that food production could support: ten naked farmers certainly would have an advantage over one naked hunter-gatherer in a fight. The other part is that neither farmers nor hunter-gatherers are naked, at least not figuratively. Farmers tend to breathe out nastier germs, to own better weapons and armor, to own more-powerful technology in general, and to live under centralized governments with literate elites better able to wage wars of conquest. Hence the next four chapters will explore how the ultimate cause of food production led to the proximate causes of germs, literacy, technology, and centralized government.

The links connecting livestock and crops to germs were unforgettably illustrated for me by a hospital case about which I learned through a physician friend. When my friend was an inexperienced young doctor, he was

called into a hospital room to deal with a married couple stressed-out by a mysterious illness. It did not help that the couple was also having difficulty communicating with each other, and with my friend. The husband was a small, timid man, sick with pneumonia caused by an unidentified microbe, and with only limited command of the English language. Acting as translator was his beautiful wife, worried about her husband's condition and frightened by the unfamiliar hospital environment. My friend was also stressed-out from a long week of hospital work, and from trying to figure out what unusual risk factors might have brought on the strange illness. The stress caused my friend to forget everything he had been taught about patient confidentiality: he committed the awful blunder of requesting the woman to ask her husband whether he'd had any sexual experiences that could have caused the infection.

As the doctor watched, the husband turned red, pulled himself together so that he seemed even smaller, tried to disappear under his bedsheets, and stammered out words in a barely audible voice. His wife suddenly screamed in rage and drew herself up to tower over him. Before the doctor could stop her, she grabbed a heavy metal bottle, slammed it with full force onto her husband's head, and stormed out of the room. It took a while for the doctor to revive her husband and even longer to elicit, through the man's broken English, what he'd said that so enraged his wife. The answer slowly emerged: he had confessed to repeated intercourse with sheep on a recent visit to the family farm; perhaps that was how he had contracted the mysterious microbe.

This incident sounds bizarrely one-of-a-kind and of no possible broader significance. In fact, it illustrates an enormous subject of great importance: human diseases of animal origins. Very few of us love sheep in the carnal sense that this patient did. But most of us platonically love our pet animals, such as our dogs and cats. As a society, we certainly appear to have an inordinate fondness for sheep and other livestock, to judge from the vast numbers of them that we keep. For example, at the time of a recent census, Australia's 17,085,400 people thought so highly of sheep that they kept 161,600,000 of them.

Some of us adults, and even more of our children, pick up infectious diseases from our pets. Usually they remain no more than a nuisance, but a few have evolved into something far more serious. The major killers of humanity throughout our recent history—smallpox, flu, tuberculosis, malaria, plague, measles, and cholera—are infectious diseases that evolved

from diseases of animals, even though most of the microbes responsible for our own epidemic illnesses are paradoxically now almost confined to humans. Because diseases have been the biggest killers of people, they have also been decisive shapers of history. Until World War II, more victims of war died of war-borne microbes than of battle wounds. All those military histories glorifying great generals oversimplify the ego-deflating truth: the winners of past wars were not always the armies with the best generals and weapons, but were often merely those bearing the nastiest germs to transmit to their enemies.

The grimmest examples of germs' role in history come from the European conquest of the Americas that began with Columbus's voyage of 1492. Numerous as were the Native American victims of the murderous Spanish conquistadores, they were far outnumbered by the victims of murderous Spanish microbes. Why was the exchange of nasty germs between the Americas and Europe so unequal? Why didn't Native American diseases instead decimate the Spanish invaders, spread back to Europe, and wipe out 95 percent of Europe's population? Similar questions arise for the decimation of many other native peoples by Eurasian germs, as well as for the decimation of would-be European conquistadores in the tropics of Africa and Asia.

Thus, questions of the animal origins of human disease lie behind the broadest pattern of human history, and behind some of the most important issues in human health today. (Think of AIDS, an explosively spreading human disease that appears to have evolved from a virus resident in wild African monkeys.) This chapter will begin by considering what a "disease" is, and why some microbes have evolved so as to "make us sick," whereas most other species of living things don't make us sick. We'll examine why many of our most familiar infectious diseases run in epidemics, such as our current AIDS epidemic and the Black Death (bubonic plague) epidemics of the Middle Ages. We'll then consider how the ancestors of microbes now confined to us transferred themselves from their original animal hosts. Finally, we'll see how insight into the animal origins of our infectious diseases helps explain the momentous, almost one-way exchange of germs between Europeans and Native Americans.

NATURALLY, WE'RE DISPOSED to think about diseases just from our own point of view: what can we do to save ourselves and to kill the

microbes? Let's stamp out the scoundrels, and never mind what *their* motives are! In life in general, though, one has to understand the enemy in order to beat him, and that's especially true in medicine.

Hence let's begin by temporarily setting aside our human bias and considering disease from the microbes' point of view. After all, microbes are as much a product of natural selection as we are. What evolutionary benefit does a microbe derive from making us sick in bizarre ways, like giving us genital sores or diarrhea? And why should microbes evolve so as to kill us? That seems especially puzzling and self-defeating, since a microbe that kills its host kills itself.

Basically, microbes evolve like other species. Evolution selects for those individuals most effective at producing babies and at helping them spread to suitable places to live. For a microbe, spread may be defined mathematically as the number of new victims infected per each original patient. That number depends on how long each victim remains capable of infecting new victims, and how efficiently the microbe is transferred from one victim to the next.

Microbes have evolved diverse ways of spreading from one person to another, and from animals to people. The germ that spreads better leaves more babies and ends up favored by natural selection. Many of our "symptoms" of disease actually represent ways in which some damned clever microbe modifies our bodies or our behavior such that we become enlisted to spread microbes.

The most effortless way a germ could spread is by just waiting to be transmitted passively to the next victim. That's the strategy practiced by microbes that wait for one host to be eaten by the next host: for instance, salmonella bacteria, which we contract by eating already infected eggs or meat; the worm responsible for trichinosis, which gets from pigs to us by waiting for us to kill the pig and eat it without proper cooking; and the worm causing anisakiasis, with which sushi-loving Japanese and Americans occasionally infect themselves by consuming raw fish. Those parasites pass to a person from an eaten animal, but the virus causing laughing sickness (kuru) in the New Guinea highlands used to pass to a person from another person who was eaten. It was transmitted by cannibalism, when highland babies made the fatal mistake of licking their fingers after playing with raw brains that their mothers had just cut out of dead kuru victims awaiting cooking.

Some microbes don't wait for the old host to die and get eaten, but

instead hitchhike in the saliva of an insect that bites the old host and flies off to find a new host. The free ride may be provided by mosquitoes, fleas, lice, or tsetse flies that spread malaria, plague, typhus, or sleeping sickness, respectively. The dirtiest of all tricks for passive carriage is perpetrated by microbes that pass from a woman to her fetus and thereby infect babies already at birth. By playing that trick, the microbes responsible for syphilis, rubella, and now AIDS pose ethical dilemmas with which believers in a fundamentally just universe have had to struggle desperately.

Other germs take matters into their own hands, figuratively speaking. They modify the anatomy or habits of their host in such a way as to accelerate their transmission. From our perspective, the open genital sores caused by venereal diseases like syphilis are a vile indignity. From the microbes' point of view, however, they're just a useful device to enlist a host's help in inoculating microbes into a body cavity of a new host. The skin lesions caused by smallpox similarly spread microbes by direct or indirect body contact (occasionally very indirect, as when U.S. whites bent on wiping out "belligerent" Native Americans sent them gifts of blankets previously used by smallpox patients).

More vigorous yet is the strategy practiced by the influenza, common cold, and pertussis (whooping cough) microbes, which induce the victim to cough or sneeze, thereby launching a cloud of microbes toward prospective new hosts. Similarly, the cholera bacterium induces in its victim a massive diarrhea that delivers bacteria into the water supplies of potential new victims, while the virus responsible for Korean hemorrhagic fever broadcasts itself in the urine of mice. For modification of a host's behavior, nothing matches rabies virus, which not only gets into the saliva of an infected dog but drives the dog into a frenzy of biting and thus infecting many new victims. But for physical effort on the bug's own part, the prize still goes to worms such as hookworms and schistosomes, which actively burrow through a host's skin from the water or soil into which their larvae had been excreted in a previous victim's feces.

Thus, from our point of view, genital sores, diarrhea, and coughing are "symptoms of disease." From a germ's point of view, they're clever evolutionary strategies to broadcast the germ. That's why it's in the germ's interests to "make us sick." But why should a germ evolve the apparently self-defeating strategy of killing its host?

From the germ's perspective, that's just an unintended by-product (fat consolation to us!) of host symptoms promoting efficient transmission of

microbes. Yes, an untreated cholera patient may eventually die from producing diarrheal fluid at a rate of several gallons per day. At least for a while, though, as long as the patient is still alive, the cholera bacterium profits from being massively broadcast into the water supplies of its next victims. Provided that each victim thereby infects on the average more than one new victim, the bacterium will spread, even though the first host happens to die.

SO MUCH FOR our dispassionate examination of the germ's interests. Now let's get back to considering our own selfish interests: to stay alive and healthy, best done by killing the damned germs. One common response of ours to infection is to develop a fever. Again, we're used to considering fever as a "symptom of disease," as if it developed inevitably without serving any function. But regulation of body temperature is under our genetic control and doesn't just happen by accident. A few microbes are more sensitive to heat than our own bodies are. By raising our body temperature, we in effect try to bake the germs to death before we get baked ourselves.

Another common response of ours is to mobilize our immune system. White blood cells and other cells of ours actively seek out and kill foreign microbes. The specific antibodies that we gradually build up against a particular microbe infecting us make us less likely to get reinfected once we become cured. As we all know from experience, there are some illnesses, such as flu and the common cold, to which our resistance is only temporary; we can eventually contract the illness again. Against other illnesses, though—including measles, mumps, rubella, pertussis, and the now defeated smallpox—our antibodies stimulated by one infection confer lifelong immunity. That's the principle of vaccination: to stimulate our antibody production without our having to go through the actual experience of the disease, by inoculating us with a dead or weakened strain of microbe.

Alas, some clever microbes don't just cave in to our immune defenses. Some have learned to trick us by changing those molecular pieces of the microbe (its so-called antigens) that our antibodies recognize. The constant evolution or recycling of new strains of flu, with differing antigens, explains why your having gotten flu two years ago didn't protect you

against the different strain that arrived this year. Malaria and sleeping sickness are even more slippery customers in their ability rapidly to change their antigens. Among the slipperiest of all is AIDS, which evolves new antigens even as it sits within an individual patient, thereby eventually overwhelming his or her immune system.

Our slowest defensive response is through natural selection, which changes our gene frequencies from generation to generation. For almost any disease, some people prove to be genetically more resistant than are others. In an epidemic those people with genes for resistance to that particular microbe are more likely to survive than are people lacking such genes. As a result, over the course of history, human populations repeatedly exposed to a particular pathogen have come to consist of a higher proportion of individuals with those genes for resistance—just because unfortunate individuals without the genes were less likely to survive to pass their genes on to babies.

Fat consolation, you may be thinking again. This evolutionary response is not one that does the genetically susceptible dying individual any good. It does mean, though, that a human population as a whole becomes better protected against the pathogen. Examples of those genetic defenses include the protections (at a price) that the sickle-cell gene, Tay-Sachs gene, and cystic fibrosis gene may confer on African blacks, Ashkenazi Jews, and northern Europeans against malaria, tuberculosis, and bacterial diarrheas, respectively.

In short, our interaction with most species, as exemplified by hummingbirds, doesn't make us or the hummingbird "sick." Neither we nor hummingbirds have had to evolve defenses against each other. That peaceful relationship was able to persist because hummingbirds don't count on us to spread their babies or to offer our bodies for food. Hummingbirds evolved instead to feed on nectar and insects, which they find by using their own wings.

But microbes evolved to feed on the nutrients within our own bodies, and they don't have wings to let them reach a new victim's body once the original victim is dead or resistant. Hence many germs have had to evolve tricks to let them spread between potential victims, and many of those tricks are what we experience as "symptoms of disease." We've evolved countertricks of our own, to which the germs have responded by evolving counter-countertricks. We and our pathogens are now locked in an escalat-